6. TEMPORAL TRENDS

6.1. INTRODUCTION

Small amounts of dioxin-like compounds may be formed during natural fires, suggesting that these compounds may have always been present in the environment. However, it is generally believed that greater amounts of these compounds have been produced and released into the environment in association with human industrial and combustion practices. As a result, environmental levels are likely to be higher in modern times than in earlier times. The trend of increasing levels may now be reversing (i.e., releases and environmental levels may be gradually decreasing), however, with changes in industrial practices. As discussed in Volume II, the potential for environmental releases of dioxin-like compounds has been reduced as a result of the switch to unleaded automobile fuels and associated use of catalytic converters and reduction in halogenated scavenger fuel additives (remaining uses of leaded fuel include chain saws, logging machinery, and mowers), process changes at pulp and paper mills, improved emission controls for incinerators, and reductions in the manufacture and use of chlorinated phenolic intermediates and products.

This chapter describes trends in the levels of dioxin-like compounds that have been observed in various environmental media and foods, as well as evidence of downward trends in exposure to dioxin-like compounds in humans. The downward trend in human exposure is supported by a modeling exercise that reconstructs the most likely past doses of dioxin-like compounds contributing to observed body burdens. Reviews of several studies and the modeling exercise are followed by several observations with regard to temporal trends of dioxin-like compounds.

6.2. SEDIMENT CORE STUDIES OF TEMPORAL TRENDS

Questions regarding the contribution made by natural sources to the overall environmental burden of CDDs and CDFs can be partially addressed using the results from analyses of the temporal distribution of CDD/CDFs in sediment core samples. Sediment cores provide a historical record of contaminant inputs into the environment and have been used by several researchers to study temporal trends in CDD/CDF deposition. Studies at

various sites in the United States and Europe suggest that environmental concentrations of CDD/CDFs began to increase rapidly in the 1930s and peaked around the 1970s.

Czuczwa and Hites (1984) analyzed sediment core samples taken in Lake Huron by the University of Michigan's Great Lakes Research Station. Sedimentation rates within the core samples were determined using Cs-137 and Pb-210 techniques. Those rates were used as a basis for relating depth of core sample to time of deposition. CDD/CDFs were detected in the core samples with no appreciable degradation over time. The most abundant CDD/CDFs were OCDDs and HpCDD/CDFs. Analysis of sample depth showed that the concentration of CDD/CDFs increased steadily beginning in approximately 1940 and leveled off around 1960. Correlations were observed over time between the levels of CDDs and CDFs in the sediment cores and the total volume of synthetic chloro-aromatics produced by the petrochemical industry in the United States. However, coal consumption did not show a good correlation with CDD/CDF concentrations over time. Czuczwa and Hites (1984) concluded that the history of sedimentation rates of CDD/CDFs in core samples from Lake Huron reflected of atmospheric deposition from the combustion of synthetic chloro-aromatics.

In a similar study, Czuczwa et al. (1985a) reported on the temporal variability of CDD/CDFs in sediment core samples taken from a wilderness lake, located in an uninhabited and undeveloped island (Siskiwit Lake, Isle Royale) in Lake Superior. The only mechanism of contaminant input into the lake was believed to be atmospheric transport and deposition. The historical record of CDD/CDF concentration in the core samples showed that CDD/CDFs were virtually absent from the sediments until around 1940. All CDD/CDF homologue groups were detected in sediment samples near the surface, with HpCDDs and OCDDs accounting for the highest percentage of total CDD/CDFs. Comparisons were made between the congener profiles found in the lake sediments and congener profiles found in urban air particles. A correlation coefficient of 0.997 was observed, leading to the conclusion that CDD/CDFs entered the lake system from aerial transport and deposition.

Smith et al. (1992, 1993) analyzed sediment core layers from Green Lake, located near Syracuse, New York, to determine temporal trends in the deposition of CDDs and CDFs since the beginning of the industrial era (circa 1860). This deep lake (200-foot depth) is thought to be affected only by atmospheric deposition because no industrial

inputs are present and motorboats are not allowed. Relatively low concentrations of CDDs and CDFs (10 pg/g or less) were observed in sediments deposited from 1860 to 1930. However, concentrations increased rapidly thereafter, reaching a peak in the mid-1960s when total CDD concentrations exceeded 1,300 pg/g and total CDF concentrations exceeded 250 pg/kg. The concentrations of CDDs and CDFs have declined rapidly since the mid-1960s and, in 1986–1990, were measured at 750 pg/g as total CDD/CDF. In the most recent samples, HpCDD and OCDD dominated the mixture of CDD/CDFs. This observation is consistent with that of Czuczwa et al. (1985a). The authors speculated that the decline in CDD/CDF concentrations over time may be due to the switch to unleaded fuels for vehicles.

Sediment cores from the Hudson River were analyzed by Smith et al. (1995). The results indicated that the subsurface sediment layers, dated between 1950 and 1980, had the highest concentrations of CDD/CDFs. OCDD, HpCDF, and OCDF accounted for the highest percentage of total CDD/CDFs in these cores. Pearson et al. (1995) studied sediment cores collected from the Great Lakes (i.e., Lakes Superior, Michigan, and Ontario) and remote inland lakes. The researchers calculated CDD/CDF accumulation rates as the product of concentration (pg/g) and sedimentation rate (g/cm^2-yr). The results of this study indicated that CDD/CDF accumulation began in the 1930s–1940s and peaked in the early to mid-1970s. Lake Ontario (350 to 575 pg/cm^2-yr) and Lake Michigan (25 to 100 pg/cm^2-yr) were found to have higher CDD/CDF accumulation rates than Lake Superior and the remote inland lakes (5 to 10 pg/cm^2-yr). CDD/CDF profiles for these lakes indicated that OCDD dominated. However, the homologue profile for Lake Ontario differed from the other lakes, leading the authors to speculate that different nonatmospheric sources were responsible for the CDD/CDFs found in this lake. MacDonald et al. (1992) observed similar temporal results in Canada. MacDonald et al. (1992) observed that OCDD and PCB-77 concentrations in sediments collected from the Strait of Georgia, British Columbia, began to increase in about 1940, reaching a maximum in about 1970. However, 2,3,7,8-TCDF concentrations did not begin to increase until about 1965 as a result of discharges of chlorine bleach effluent from local pulp mills.

Lebeuf et al. (1995) observed decreasing dioxin-like PCB (i.e., PCBs 77, 126, and 169) trends in sediments from the Lower Estuary and Gulf of St. Lawrence, Canada. Two sediment cores were collected from sites approximately 50 km apart and sliced into 25

samples. PCB concentrations were found to increase with increasing depth. These results indicate that recent inputs to this water body have decreased substantially (Lebeuf et al., 1995).

Rappe et al. (1997) analyzed sediment core samples from five lakes in southern Mississippi. The sediment cores were collected from five man-made recreational lakes with no known industrial point source of CDD/CDFs and low atmospheric deposition rates. Cores were subdivided into sections to evaluate temporal trends in deposition of CDD/CDFs. No observable trend for levels of CDDs, homologues, or I-TEQs correlating to the age of the strata could be identified.

Recently, EPA/DOE conducted a time-trend study of dioxin-like compounds in sediment cores (Cleverly et al., 1996; Versar, 1996). Cores from 11 lakes/reservoirs were collected, sectioned, and dated using ^{137}Cs and ^{210}Pb dating techniques, and analyzed for CDD/CDFs and PCBs. The lakes were located in various geographic locations throughout the United States (10 within the continental United States and 1 in Arctic/Alaska) and were selected to represent background conditions (i.e., no known CDD/CDF sources). For several of the lakes, dated samples were available for time periods ranging from the 1700s to the present. The results of the study indicated that CDD/CDF and PCB inputs to U.S. lakes have increased over time, with significant increases occurring after the 1930s. This is consistent with the findings of other researchers. In general, a minimum of one order of magnitude increase in concentration occurred from the pre- to post-1930s timeframe. With few exceptions, this observation was consistent for all 2,3,7,8-substituted CDD/CDF congeners, CDD/CDF homologue groups, and PCBs. The observed temporal trends were consistent across lakes (except the Arctic/Alaska lake), especially for lakes within the same geographic region. For some lakes, a downward trend appeared to exist for the most recent periods. The point of inflection for this downward trend varied across lakes, but appeared to occur between the 1950s and 1970s. Figure 6-1 depicts the changes in concentration over time for Beaver Lake, Washington. The data also indicate that the CDD/CDF and PCB profiles in these lakes were similar across all periods and across all lakes. This may suggest that the relative congener-specific inputs from the atmosphere have remained consistent over time and are similar across all geographic regions. Relationships also were observed between CDD/CDF and PCB trends and indicators of anthropogenic activities (i.e., PCP production, leaded gasoline sales, carbon monoxide

emissions, and PCB releases) that may be associated with production of CDD/CDFs or PCBs. However, it should be noted that correlations between these variables do not necessarily reflect causal relationships.

Several studies have also evaluated sediment cores from European lakes. Czuczwa et al. (1985b) studied temporal trends in three Swiss lakes (Lakes Zurich, Lugano, and Baldegg). No CDD/CDFs were detected in the sediments prior to 1945, but increasing levels were observed in subsequent time periods. The most abundant congeners found in the uppermost sediment samples were OCDD, HpCDD/CDFs, and OCDF. Using sedimentation rates and CDD/CDF surface sediment concentrations, the estimated accumulation rates were 300 pg/cm^2-yr for Lake Zurich, 270 pg/cm^2-yr for Lake Lugano, and 190 pg/cm^2-yr for Lake Baldegg. The authors noted that the similarities in congener profiles and fluxes of CDD/CDF in Switzerland and the United States may be indicative of similar sources of CDD/CDFs. Similar trends were noted by Beurskens et al. (1993, 1994). These researchers evaluated sediment cores from Lake Ketelmeer, a sedimentation area of the Rhine River in The Netherlands. CDD/CDF concentrations were shown to increase after the 1940s and peak between 1960 and 1980. Similar results were obtained for coplanar PCBs.

Sediment cores from two lakes in the Black Forest region of Germany were analyzed for temporal trends in CDD/CDF deposition by Schramm et al. (1994). CDD/CDFs were found to have increased by a factor of 13 since the 1930s. Recently, Hagenmaier and Walczok (1996) evaluated CDD/CDF levels in dated sediment core samples collected from Lake Constance, Germany. Cores were collected in December 1995 and April 1996. Based on a preliminary dating scheme, the results indicated that the I-TEQ$_{DF}$ concentrations for these samples began increasing around 1940, reached their peak around 1970 to 1975, and then began decreasing. The homologue profiles for the 1940s sediments were similar to those in recent deposition samples.

Using dated sediment core analyses, Alcock et al. (1997a) reported that CDD/CDF concentrations in a remote lake in Scotland began increasing in the 1860s and 1870s and peaked in the 1950s and 1960s. According to Alcock et al. (1997a), concentrations appear to have decreased in recent years. Brzuzy and Hites (1995) reported on changes over time in the CDD/CDF homologue profile for Lake Windermere in the United Kingdom. CDFs accounted for a significant fraction of the total CDD/CDF concentration in sediment

core sections dated 1946–1950. In contrast, sections dated 1988-1992 had a significantly lower fraction of CDFs, and the profile was dominated by OCDD and HpCDD. These results provide evidence that the sources of CDD/CDF deposition have changed over time, with CDFs accounting for a much higher percentage of inputs during the earlier period.

Sediment core samples from the Baltic Proper, near Sweden, showed detectable levels of CDD/CDFs dating back as early as 1882 (Kjeller and Rappe, 1994). CDD/CDFs increased slowly in the sediment strata dated between 1882 (92 pg/g) and 1962 (233 pg/g) and then increased rapidly in the 1970s. Total CDD/CDFs were estimated to be 520 pg/g in 1970 and 1,803 pg/g in 1978. CDD/CDFs decreased to a concentration of 1,454 pg/g in the most recent layer, dated 1985. DeWit et al. (1990) also observed that CDD/CDF concentrations were higher in deeper sediment layers from the Baltic Sea. I-TEQ_{DF} concentrations in surface sediments (i.e., most recently deposited) were approximately 20 times higher than in sediments collected at depths of 22 to 28 cm.

Sediment cores were collected to a depth of 5–18 m at three locations near Osaka Bay, Japan, and analyzed for CDD/CDFs (Sakai et al., 1998). CDD/CDFs found at Yodo River, which is influenced by urban activities, increased slowly from 1980, reached a peak in 1993, and then dropped to a lower concentration before stabilizing. Comparison between the southern and northern parts of Lake Biwa, representing areas more and less affected by human activities, respectively, showed that CDD/CDF concentrations at the southern part of Lake Biwa began increasing around 1955 and increased dramatically in 1964, reaching a peak in 1973. At the northern part of Lake Biwa, CDD/CDF concentrations increased dramatically in the late 1960s and continued to increase until the 1980s, when they leveled off. Homologue profiles for the sediment core sampled from the northern part of Lake Biwa showed that OCDD was the dominant congener since 1842, followed by TCDD after 1935. The concentrations of OCDD and TCDD continued to increase after 1935. The sources of these two congeners were suspected to be herbicides, preservatives, and municipal solid waste incinerators (Sakai et al., 1998).

CDD/CDFs have also been detected in remote Arctic sediment core samples, but at very low concentrations (Tan et al., 1993; Vartiainen et al., 1995). Tan et al. (1993) analyzed a sediment core collected from Wonder Lake, Alaska. With the exception of 1,2,3,4,6,7,8-HpCDF and OCDF, which had concentrations of 13 and 15 pg/g, respectively, in all sediment sections dated between 1590 and 1790, CDD/CDF

concentrations were less than 8 pg/g. The authors suggest that these results support *de novo* synthesis of CDD/CDFs. Vartiainen et al. (1995) found total CDD/CDF levels of 2.29 pg/g in sediments dated 1890 and 55 pg/g in sediments dated 1994. OCDD dominated in these samples, followed by the hepta- and hexa-chlorinated congeners.

The results of these sediment core studies provide evidence that deposition of dioxin-like compounds began increasing dramatically after the 1930s and continued throughout the 1960s. Decreases appear to have occurred only during the most recent periods. In all of these studies, the higher chlorinated compounds dominated the homologue profiles. These observations are consistent among cores collected in various locations throughout the United States and Europe. CDD/CDFs have been observed both in relatively remote lakes, as well as in lakes close to industrialized areas. This suggests that atmospheric transport and deposition may be an important mechanism of entry into these lakes.

6.3. TEMPORAL TRENDS IN SOIL, VEGETATION, AND AIR

Temporal trends in CDD/CDF and PCB deposition have also been studied in other types of environmental media including soil, vegetation, and air samples. Kjeller et al. (1991) analyzed archived soil samples dated from 1846 to 1986 from semirural plots in the United Kingdom. Herbage samples dated from 1891 to 1988 that originated from a grassland area were also tested for CDD/CDFs (Kjeller et al., 1991, 1996). All CDD/CDF homologue groups were detected in soil and herbage samples from all time periods, and the CDD/CDF concentrations increased over time beginning at about 1900 (Kjeller et al., 1991). The concentrations of total CDDs in soil increased from 31 to 92 pg/g between 1893 and 1986. CDD/CDFs in the vegetation samples remained essentially constant between 1861 and 1945, increased to peak levels in the early 1960s, declined, and then reached a second peak in the late 1970s (Kjeller et al., 1996). CDD/CDFs were about 7 to 8 times higher in the 1960s to 1980s than in the sample from 1891 to 1900. In the most recent sample (1991 to 1993), total CDD/CDFs declined to levels similar to those observed in pre-1946 samples (Kjeller et al., 1996).

In a similar study, Alcock et al. (1997b) presented evidence that CDD/CDFs were present in UK soils before the widespread development of chloroaromatics (around 1930s). A previously unopened bottle of soil, collected in 1881 from Rothamsted Experimental

Station as part of an agricultural experiment, was analyzed for CDD/CDFs. The soil sample contained 0.7 ppt I-TEQ_{DF}, with OCDD, 1,2,3,4,6,7,8-HpCDD, and 1,2,3,4,6,7,8-HpCDF as the dominant congeners. Great care was taken to avoid contamination with modern air and dust for the initial analysis, and the sample was subsequently exposed to laboratory air for 32 days to determine whether current air concentrations would alter the CDD/CDF concentrations detected in the archived soil. The results indicated that such exposure did not alter the soil concentration of CDD/CDF. Modern soil samples collected from the same field plot were found to contain 1.4 ppt I-TEQ_{DF}. The authors speculated that the increase was presumably a result of cumulative atmospheric deposition of CDD/CDFs. The results also indicated that although the soils from 1881 had lower concentrations of CDD/CDFs, they contained similar congener profiles of CDD/CDF as modern soil. This may indicate long-term persistence of CDD/CDFs in soil, and similarities in source inputs over time (Alcock et al., 1997b). Alcock et al. (1997a) also summarized temporal trends in sediments, archived vegetation, soil, food groups, and direct air measurements from the United Kingdom. They suggested that concentrations in UK media were the highest between the 1950s and the 1970s and were directly related to human activities. Since the 1970s, CDD/CDF concentrations have shown a consistent decline. According to Alcock et al. (1997b), current herbage concentrations are similar to pre-1946 levels, and ambient air monitoring data from London and Manchester indicate that CDD/CDF air concentration have steadily declined since the 1970s. Archived soil, herbage, and air samples collected in the United Kingdom between 1942 and 1992 were used to evaluate changes in PCB emissions over time (Harner et al., 1995). The concentrations of PCB congeners 28, 52, 138, and 153 in these samples rose from near zero in 1935 to a maximum in the late 1960s and then fell steadily to their present levels.

Hiester et al. (1995) observed a decrease of CDD/CDF concentrations in Germany's ambient air over a 6-year period. Ambient air samples were collected over 12 sampling intervals from four sites in the heavily industrialized Rhine-Ruhr region of Germany during 1987–1988 and 1993–1994, and analyzed for CDD/CDFs. Total I-TEQ_{DF}s for these sites ranged from 0.13 pg/m^3 to 0.33 pg/m^3 during 1987–1988, and from 0.04 pg/m^3 to 0.12 pg/m^3 during 1993–1994. Reductions in CDD/CDF I-TEQ_{DF}s at these sites ranged from 46 to 69 percent over the 6-year period (i.e, from 0.22 pg/m^3 to 0.13 pg/m^3 at Dortmund, and

from 0.13 pg/m^3 to 0.04 pg/m^3 at Köln). These reductions were attributed to abatement actions taken since 1989 (Hiester et al., 1995).

6.4. TEMPORAL TRENDS IN WILDLIFE

Temporal trends in CDD/CDFs and PCBs have also been studied in wildlife, including fish and bird eggs. Hebert et al. (1994) analyzed pooled herring gull eggs for CDDs annually between 1981 and 1991. The eggs were collected from colonies in the Great Lakes and the Gulf of St. Lawrence River. Analyses results indicate that CDD levels declined between 1981 and 1984, but that CDD levels have remained relatively constant since 1984. DeWit et al. (1994) evaluated temporal trends in the levels of CDD/CDFs and coplanar PCBs in the biota of Sweden. Guillemot eggs were collected from the Island of St. Karlso in the Baltic Proper (Sweden) between 1969 and 1992, and pike samples were collected from Lake Storvindeln in Lapland, Sweden, between 1968 and 1992. During these time periods, the concentrations of CDD/CDFs and PCBs decreased in both species. Roos et al. (1998) confirmed that PCB concentrations decreased significantly in the Baltic Sea between 1989 and 1997 at an annual rate of 2–4 percent by analyzing samples from 54 juvenile grey seals caught off the Swedish coast. Roos et al. (1998) also found that the PCB concentrations in herring, cod, and guillemot eggs caught from the Baltic Sea during the period 1969–1996 decreased over time by 9–10 percent annually. Decreases in PCBs since the 1970s were also observed in fish from Finland (Korhonen et al., 1995). Pike samples from both inland lakes and coastal areas of Finland had significantly lower total PCB concentrations in 1994 (1.7 to 2.1 μg/g) than in 1971 (> 7 to 10 μg/g).

U.S. EPA (1994a) reported a decline in PCB concentrations in lake trout from Lake Michigan since the late 1970s; however, PCB concentrations currently appear to be approaching equilibrium in the Great Lakes system. U.S. EPA (1994a) attributed the decline in tissue concentrations to reductions in pollutant loadings to the water column. The levels of PCBs in coho salmon also declined during the early 1980s, but have remained relatively constant since that time. According to U.S. EPA (1994a), the leveling off of PCB concentrations in the Great Lakes is related to "(1) historically contaminated sediments; (2) tributaries inputs resulting from point sources, spills, and runoff from both urban and rural areas, and resuspension from contaminated sediments; and (3) atmospheric deposition of pollutants."

Hilbert et al. (1997) analyzed cod livers collected from Danish waters between 1973 and 1996 for total PCBs (Aroclor 1260). The results indicated that PCB levels in cod livers decreased during the past three decades. Total PCBs ranged from approximately 4 to 8 mg/kg (fresh weight) in 1973 at five locations in Danish waters to less than 1 mg/kg (fresh weight) in 1996 in those same waters.

Huestis et al. (1997) examined the temporal and age-related trends of CDD/CDFs and coplanar PCBs in Lake Ontario lake trout. Archived samples of 4-year-old lake trout, collected between 1977 and 1993 from the eastern basin of Lake Ontario at Main Duck Island (MDI), were analyzed for CDD/CDF/PCBs. Three- to 9-year-old trout were collected from the western end of the basin at Port Credit. The results of the temporal trends analysis indicated that CDD/CDF/PCB concentrations were at their highest levels in 1977 and lowest in 1987. The total TEQ_{DFP}-WHO_{94} was 583 ppt in 1977, compared to 124 ppt in 1993. The most important contributor to the total TEQ_{DFP}-WHO_{94} was PCB 126. This PCB congener contributed between 40 to 50 percent of the total TEQ_{DFP}-WHO_{94}, depending on the year examined. The contribution of 2,3,7,8-TCDD accounted for 15 to 20 percent of the total TEQ_{DFP}-WHO_{94}. CDD/CDF contaminant profiles for 1977, 1982, and 1991 suggest an increase of the proportion of 2,3,7,8-TCDF as the proportion of 2,3,7,8-TCDD decreases. The study also evaluated age-related effects of CDD/CDF/PCB concentrations. The results of the analyses of 3- to 9-year-old fish indicate that as the age of the fish increases, the level of contamination also increases.

Boumphrey et al. (1998) examined 10 gannet eggs taken from Ailsa Craig, a colony in the northern part of the Irish Sea, every 2 years from 1977 to 1992 to analyze temporal trends in total PCB and PCB congener concentrations. The results indicated that the average total PCB concentration was 6.08 μg/g in 1977 and 2.5 μg/g in 1992 with an annual rate of decline of approximately 0.25 μg/g. Individual PCB congeners declined at various rates, which resulted in different congener profiles between 1977 and 1992.

6.5. TEMPORAL TRENDS IN FOOD PRODUCTS

Recent studies have evaluated trends in concentrations of dioxin-like compounds in food by analyzing levels of PCBs and CDD/CDFs in food products from different time periods. The United Kingdom's Ministry of Agriculture, Fisheries, and Food (MAFF) compared the levels of CDD/CDFs found in commercially available cows' milk in 1990 to

the levels in samples collected in 1995 (MAFF, 1997a). The 1995 samples were collected from 12 locations in England and corresponded, where possible, to the 1990 sampling locations. Analysis of the 1995 samples was performed on a pool of 105 pints of full fat milk from each location. As shown in Table 6-1, the lipid-based I-TEQ_{DF} concentrations in 1990 ranged from 1.1 to 3.3 ppt, while the 1995 I-TEQ_{DF} levels decreased to between 0.67 and 1.4 ppt. Table 6-1 also reports PCB levels for the 1995 samples. Lipid-based TEQ_P-WHO_{94} levels ranged from 0.75 to 2.2 ppt. No PCB analysis for the 1990 cows' milk samples was presented.

Fürst and Wilmers (1995) compared the levels of CDD/CDFs found in German dairy products in 1990 to the levels in 120 dairy samples collected in 1994. Over the 4-year period, mean I-TEQ_{DF} concentration in milk fat decreased by almost 25 percent from 1.35 ppt to 1.02 ppt. Similar reductions were noted in human milk fat (Fürst and Wilmers, 1995).

To examine trends in CDD/CDF and PCB concentration in American food products, Winters et al. (1998) analyzed 14 preserved food samples from various decades of the 20th century for 7 dioxin-like coplanar PCBs and the 17 2,3,7,8-substituted dioxin and furan congeners. The authors compared the concentrations found in historical samples to the current dioxin concentrations observed in the national food surveys for beef (Winters et al., 1996a; 1996b), pork (Lorber et al., 1997), poultry (Ferrario et al., 1997), and milk (Lorber et al., 1998). As shown in Table 6-2, all 10 samples, dated from 1957 to 1982, had I-TEQ_{DF} concentrations higher than the current mean concentrations (when nondetects were set to one-half the limit of detection). Similarly, mean TEQ_P-WHO_{94} concentrations were higher than current mean concentrations for 12 of the 13 samples taken between 1945 and 1983. If these samples are indicative of past CDD/CDF concentrations, normalized I-TEQ_{DF} results suggest CDD/CDF levels 2 to 3 times higher, and PCB levels over 10 times higher during the 1950s, 1960s, and 1970s than current concentrations. As shown in Figures 6-2 and 6-3, I-TEQ_{DF} and TEQ_P-WHO_{94} concentrations in food products began to increase with the 1957 sample and continued to increase throughout the 1960s and 1970s. The peak concentration was observed in the 1968 poultry sample. This trend in CDD/CDF and PCB concentrations in food products is consistent with the pattern observed in sediment cores, as discussed in Section 6.2 (Cleverly et al., 1996; Versar, 1996; Smith et al., 1992, 1993, 1995; Czuczwa et al., 1984, 1985a).

6.6. TEMPORAL TRENDS IN HUMAN EXPOSURE

Several studies have examined trends in the dietary intake of CDD/CDFs and PCBs. In 1995–1996, MAFF reported on its analysis of Total Diet Study samples collected in 1982 and 1992 from 24 locations in the United Kingdom (UK) (MAFF, 1995, 1996, 1997b). Of the 11 food groups examined, 10 groups represented the major dietary contribution to intake of dioxins and PCBs, and 1 group represented bread because it is a dietary staple. The results indicated that total dietary intake of PCBs and CDD/CDFs by consumers in the United Kingdom decreased dramatically between 1982 and 1992. Dietary intake of CDD/CDFs and PCBs for specific food items was estimated by multiplying the TEQ concentration of CDD/CDFs and PCBs in the food item (calculated by setting nondetects to the limit of detection) by the average daily intake for that food item, as estimated in the UK's National Food Consumption Survey. Total dietary intake was calculated by summing the dietary intakes for all food groups. The estimated upper bound dietary intakes of CDD/CDFs and PCBs by the average adult UK consumer in 1982 and 1992 are presented in Table 6-3. The total dietary intake of CDD/CDFs was estimated to be 240-pg/day I-TEQ_{DF} in 1982 and 69-pg/day I-TEQ_{DF} in 1992. The total dietary intake of PCBs decreased from 156-pg/day TEQ_{P}-WHO_{94} in 1982 to 46-pg/day in 1992 (MAFF, 1997b). Harrison et al. (1998) also reported on composite human milk samples collected as part of the MAFF study. Lipid-based CDD/CDF I-TEQ levels in Birmingham, England, were 37 ppt in 1987–1988 and 21 ppt in 1993–1994. I-TEQ levels were 29 ppt in Glasglow in 1987–1988 and 21 ppt in 1993–1994.

Liem et al. (1997) analyzed duplicate portions of 24-hour diet samples collected in The Netherlands in 1978, 1984 to 1985, and 1994. This study was conducted to estimate the dietary intake of CDD/CDFs and PCBs in the Dutch population 18 years of age and older to evaluate trends in dietary exposures. Dietary intake was estimated by combining the results of the chemical analyses of foods with data on consumption rates from the Dutch National Food Consumption Survey. Liem et al. (1997) reported a significant reduction of CDDs and CDFs in the diet over the three time periods. The mean daily dietary intake of CDD/CDF decreased from 4.2 pg/kg I-TEQ_{DF} in 1978 to 1.8 pg/kg I-TEQ_{DF} in 1984–1985 and 0.5 pg/kg I-TEQ_{DF} in 1994. When PCBs were included in the TEQ calculation, the daily dietary intake was 11 pg/kg TEQ_{DFP}-WHO_{94} in 1978, 4.2 pg/kg TEQ_{DFP}-WHO_{94} in 1984–1985, and 1.4 pg/kg TEQ_{DFP}-WHO_{94} in 1994. The percentage decrease in 1994

samples was consistent for all the measured CDD/CDF/PCBs. The results of this study suggest that a reduction in dietary ingestion of CDD/CDFs and PCBs occurred in The Netherlands beginning in the late 1970s.

In a study similar to the United Kingdom's MAFF (1995, 1996, 1997b) study, Fürst and Wilmers (1997) found that CDD/CDF dietary levels also dropped in Germany in recent years. Several hundred food samples were randomly collected and analyzed for CDD/CDFs during 1989 and 1995. Fish products showed the greatest decline in CDD/CDF food concentrations over this period. Significant decreases also were noted for meats. Samples of more than 300 dairy products were collected in 1990 and 1994 from several dairies in North Rhine and Westphalia and analyzed for CDD/CDFs. The results indicate that from 1990 to 1994, CDD/CDF levels in cows' milk decreased approximately 25 percent. To test whether the reduction of CDD/CDF concentrations in food had a positive effect on human exposure, dietary intake of CDD/CDFs was estimated using the results of the food sample analysis described above and standard food consumption data. This analysis indicated that in the past few years, CDD/CDF intake by humans decreased by approximately 50 percent. The current average daily intake is estimated to be 69.6 pg I-TEQ_{DF} compared to a daily average intake of 127.3 pg I-TEQ_{DF} in 1990. This decrease in daily intake was also reflected in a decrease in breastmilk concentrations (Fürst and Wilmers, 1997). A study of more than 1,000 individual breastmilk samples from the North Rhine-Westphalia region showed a decrease from 34 ppt I-TEQ_{DF} in milk fat in 1989 to 14.2 ppt I-TEQ_{DF} in milk fat in 1996. This represents a 60 percent reduction since 1989.

Studies that may be used to assess temporal trends in the human body burden of dioxins and furans are extremely limited. The use of indirect exposure assessment techniques for detecting temporal trends is difficult because large-scale, long-term, nationally representative environmental monitoring for dioxins and furans has not been conducted. Short-term studies are generally not comparable because of differences in sampling protocols and analytical techniques. A potentially useful study for evaluating changes in human exposure over time is EPA's National Human Adipose Tissue Survey (NHATS), conducted between 1970 and the late 1980s. The purpose of NHATS was to monitor the human body burden of selected chemicals in the general U.S. population (U.S. EPA, 1991). NHATS used direct measurement techniques to estimate exposures. Nationwide samples of adipose tissue were collected from surgical patients and autopsied

cadavers and analyzed annually. In 1982, broadscan analysis of composited adipose tissue specimens revealed that chlorinated dioxins and furans could be detected and quantified in the U.S. population across all geographic regions and age groups (U.S. EPA, 1986). Analysis of NHATS specimens again in 1987 made temporal comparisons possible.

EPA performed statistical analyses of the 1982 and 1987 data to determine if significant differences existed between the concentrations of these compounds adipose tissue specimens. Table 6-4 presents the estimated national average concentrations for the two periods and the relative changes from 1982 to 1987. The estimated concentrations of 1,2,3,7,8-PCDD; 2,3,4,7,8-PCDF; and HxCDD in human adipose tissue were significantly lower in 1987 than in 1982 (U.S. EPA, 1991). Similar survey designs were used in the two studies, but in 1987, some changes in the analytical methods were made that may account for some of the differences in estimated concentrations. These changes included lower limits of detection and the use of additional internal quantitation standards that provided more accurate measurements. The levels of 2,3,7,8-TCDD; 1,2,3,4,6,7,8-HpCDD; and OCDD were also lower in 1987 than in 1982, but the differences were not statistically significant. No statistical comparisons were possible for 2,3,7,8-TCDF; HxCDF; 1,2,3,4,6,7,8-HpCDF; or OCDF because one or both of the annual estimates were based on data that did not meet the minimum criteria for statistical modeling (i.e., the chemical was not detected in at least 50 percent of the composites analyzed, and/or fewer than 30 composite samples were analyzed in each year). The results of this study indicate that exposure to certain dioxins and furan congeners may have decreased over this 5-year period. However, further studies are needed to verify that these changes are not a result of protocol changes, but are actual reductions in exposures.

Schecter (1991) analyzed liver tissues estimated to be 100 to 400 years old recovered from the frozen bodies of two Native American (Eskimo) women. The women died in their igloo in Point Barrow, Alaska, when they were trapped and frozen by an ice overflow. Oil was used for cooking and heating, and ventilation was poor. One woman had soot-laden lungs. The results indicated that dioxin levels were much lower in these ancient tissues than in livers of people currently living in industrial areas. Tong et al. (1990), as cited in Schecter (1991), found a lipid-based total I-TEQ_{DF} level of 0.24 ppt in one of the ancient liver samples. I-TEQ levels (i.e., CDD, CDF, and total) were nondetectable in the other ancient sample. Analysis of two liver samples from modern

times showed lipid-based total I-TEQ_{DF} levels of 13.3 ppt (Ryan et al., 1986, as cited in Schecter, 1991).

Recently, Päpke et al. (1997) analyzed 180 whole blood samples collected in Germany in 1996 for CDD/CDFs. The samples were taken only from individuals who had no known exposure to CDD/CDFs (i.e., their only exposure would be through food ingestion). The results of this study were compared to a similar study conducted in 1994. The results indicated that concentrations declined from 19.1 ppt I-TEQ_{DF} in blood lipids in 1994 to 16.5 ppt I-TEQ_{DF} in blood lipids in 1996. These results suggest human exposure to CDD/CDFs have decreased over the past several years.

Wittsiepe et al. (1998) measured CDD/CDFs in 507 blood samples collected in Germany between 1991 and 1996. The samples were intended to represent the general population (i.e., the individuals were not exposed to dioxin-like compounds as a result of occupational or accidental contacts). The results indicated that blood levels of CDD/CDFs declined significantly between 1991 and 1996, with the more recent concentrations representing approximately one-half the earlier concentrations. The mean lipid-based total CDD/CDF concentrations were 71.8 pg/g in 1991 and 334.4 pg/g in 1996 (Table 6-5).

Liem et al. (1996) detected downward trends in the levels of CDD/CDFs in human breastmilk between 1987–1988 and 1992–1993. Breastmilk samples were collected from women in 11 countries as part of a World Health Organization (WHO)-coordinated exposure study. Protocols were developed to ensure that the samples collected during the two periods were comparable. The protocols included criteria for selection of donors, sampling areas, etc. The samples were analyzed for the 17 CDD/CDF congeners, as well as 6 marker PCBs (IUPAC numbers 28, 51, 101, 138, 153, and 180). The results indicated that CDD/CDF levels are decreasing in some countries (Table 6-6). Liem et al. (1996) estimated an overall annual CDD/CDF decrease of 7.2 percent, based on the data from those countries.

In a similar study, Schecter et al. (1997) assessed CDD/CDF concentrations in blood and breastmilk samples collected in Germany and the United States during two time periods. More than 100 blood samples were collected in Germany during the years 1989 and 1994 from "persons for whom there was concern about dioxin exposure but abnormal dioxin blood levels were not found." American blood samples were collected in 1984–1989 (male veterans with no dioxin abnormalities) and 1996. The German milk

samples were collected in Westphalia in 1991 and 1995, and American samples were two pooled samples from 1988 (Binghamton, NY, and Los Angeles, CA), five individual analyses from the period 1995-1996 (Binghamton, NY), and one pooled sample from 1997 (Binghamton, NY). The results of the study indicated that from 1989 to 1994, the CDD/CDF concentrations in German blood declined from 43 ppt I-TEQ_{DF} to 19 ppt I-TEQ_{DF}. CDD/CDF concentrations in United States blood samples declined from 28 ppt I-TEQ_{DF} in 1984–1989 to 25 ppt I-TEQ_{DF} in 1996. The results of the German breastmilk analyses indicated a decline from 23 ppt I-TEQ_{DF} in 1991 to 16 ppt I-TEQ_{DF} in 1995. American milk samples showed a reduction from 17 ppt I-TEQ_{DF} in 1988 to 9 ppt I-TEQ_{DF} in 1995–1997.

Kiviranta et al. (1998) measured the concentrations of the 17 toxic CDD/CDF congeners and 6 PCB congeners (IUPAC 28, 52, 101, 138, 153, and 180) in human milk samples from primiparae mothers in Finland. Samples were collected from women in both rural and urban areas between 1992 and 1994 and compared to data from 1987 (Table 6-7). Total lipid-based CDD/CDF concentrations declined from 339 ppt (n = 37) to 217 ppt (n = 28) in rural areas between 1987 and 1992–1994; urban concentrations were similar: 375 ppt (n = 47) and 381 ppt (n = 14) for the two sample periods, respectively. I-TEQ_{DF} values decreased from 20.1 ppt to 13.6 ppt in rural areas, and 26.3 ppt to 19.9 ppt for urban areas between 1987 and 1992–1994. Total lipid-based PCB concentrations declined from 396 ng/g (n = 37) to 198 ng/g (n = 28) in rural areas, and from 496 ng/g (n = 47) to 296 ng/g (n = 14) in urban areas for the same two sample periods.

6.7. A MODELING EFFORT TO RECONSTRUCT PAST DOSES OF 2,3,7,8-TCDD

Previous sections in this chapter describe evidence supporting temporal trends in environmental concentrations and human exposure to CDD/CDF/PCBs. Levels of dioxin-like compounds appeared to increase in the environment starting from the 1930s through the 1960s, and loadings began to decline perhaps starting in the 1970s to the present. Recent evidence collected on animal food products in the United States (Winters et al., 1998), combined with body burden data, are the best evidence that human exposures to dioxins may have followed the same trends. (See Sections 6.5 and 6.6.) This section describes a third way of evaluating past exposures to dioxins. Pinsky and Lorber (1998) described an effort to statistically reconstruct the pattern of past human exposure to the most toxic dioxin congener, 2,3,7,8-TCDD (abbreviated TCDD), through use of a simple

pharmacokinetic (PK) model that included a time-varying TCDD exposure dose. This section summarizes the procedure and presents some key results from this modeling exercise. The original reference (Pinsky and Lorber, 1998) should be obtained for further detail.

A first-order, one-compartment PK model was used to compute an individual's body lipids' TCDD concentration over time. Key inputs for that model include: (1) a time-varying dose of TCDD (expressed in units of pg/kg-day), (2) a fraction of dose absorbed into the body lipid compartment (assumed to be constant), (3) the volume of the body lipid compartment (assumed to be time varying), and (4) a rate of TCDD loss from the lipid compartment (modeled as a function of the percent of body fat). To calculate the rate of TCDD loss, a model was needed to predict how body lipid volumes vary over time, in addition to a model of how overall body weight varied over time.

In this modeling exercise, all inputs were fixed except the time-varying dose of TCDD. Using Bayesian statistical approaches, the dose was "calibrated" to best fit a set of data on TCDD concentration in body lipids. These data, shown in Table 6-8, were obtained from studies that focused on persons with no known direct exposure to dioxins and, as such, measured background exposure levels. In terms of this modeling exercise, the most important data from this set, were from the 1970s, suggesting that body lipid concentrations of TCDD were above 10 ppt during those years (VA/U.S. EPA, 1988). Current data from the 1980s into the 1990s show TCDD concentrations below 10 ppt (U.S. EPA, 1991; Michalek et al., 1998; Andrews et al., 1989).

The feature of the Bayesian approach that is most relevant to this calibration modeling exercise was the use of constraints on the input functions. In other words, much of the evidence described earlier in this chapter suggests an expected trend on the dose function that was being calibrated in this modeling exercise (i.e., that the TCDD dose may have increased from the 1930s to the 1970s and declined thereafter). An examination of the existing trend data suggests, specifically, the following for the current dose modeling purposes: (1) a peak in environmental levels appears to have occurred in the 1960s or 1970s, (2) early century levels are from 2 to >33 times lower than the peak, (3) late 1980s levels are from 1 to 20 times lower than the peak, and (4) late 1980s levels are higher than early century levels; in all cases, the ratio of peak to 1980s levels is lower than the ratio of peak to early century levels. Also, and importantly for this modeling exercise,

the estimate of TCDD exposure dose based on the 1994 release of this dioxin reassessment document (U.S. EPA, 1994b) was 0.17 pg TCDD/kg-day. Using these trends, the following Bayesian "plausibility criteria" were established for calibration modeling purposes:

1. A range of 0.0 to 0.50 pg TCDD/kg-day for the exposure dose in 1990. The same plausible range was used for the 1900 dose.
2. Ranges of 2 to 200 for the ratio of peak to 1900 dose, and 1 to 100 for peak to 1990 dose.
3. Peak year set between 1945 and 1980.

Finally, to ensure a smooth exposure curve, a limit of 20 percent was set on the rate of decrease from the peak exposure level going forward or backward 1 year.

Pinsky and Lorber (1998) detailed how well the calibrated doses duplicated the measured body burdens shown in Table 6-8. In general, several slightly different calibrated dose functions fit the data equally well. An example of a family of similar dose curves is shown in Figure 6-4. In that figure, the dose appears to increase from the 1940s through the 1960s, then begins to drop through the 1970s, with a baseline level being reached by the 1980s. This qualitatively fits some of the trend data described previously. However, while the calibrated model fits the data reasonably well, it does not fit the data perfectly. Obviously, the lack of a perfect model fit beginning in the 1970s could be partially attributed to the lack of observed body concentrations of 2,3,7,8-TCDD. As seen in Figure 6-4, the dose curves appear to converge after the 1970s, when there were observed data, while the dose curves seem to diverge prior to 1970, when there were no data on which to base the calibration. Also, some data were inconsistent, particularly the National Human Adipose Tissue Study (NHATS) data from 1982 and 1987 (U.S. EPA, 1991). Specifically, in a comparison of the NHATS 1982 and 1987 data, the mean TCDD concentration increased considerably from 1982 to 1987 in the oldest age group (45+), but decreased considerably in the two younger age groups. Further, the NHATS 1982 data do not display the trend of increasing TCDD concentrations by age that is seen in most other studies done in the 1980s (mean was 6.9 pg/g in the 15–44 age group and 5.5 pg/g in the 45+ age group), while the age trend in NHATS 1987 data (mean of 4.4 pg/g in the 15–44 age

group versus 9.4 pg/g in the 45+ age group) seems exaggerated. These trends are difficult to explain with the current modeling structure; subsequently, all models with a good data to model fit overpredicted the 1982 mean and underpredicted the 1987 mean in the highest age group. More data from prior to and during the 1970s would have provided a much more useful database from which to calibrate the model; however, no other TCDD body concentration data could be found from those time periods.

Despite the lack of a perfect fit of the model to the data, several informative findings resulted from this exercise.

1. The model calibration exercise was regenerated using two changes to the initial Bayesian constraints on the shape of the exposure/dose curve. One change essentially dropped all constraints in order to test whether the imposition of the constraints restricted how well a calibrated dose curve could fit the data. It was found that a best-fit solution for exposure with no constraints provided only an insignificant improvement. The second change was to constrain the exposure dose, making it constant over time. Results showed that the temporally varying dose provided a significantly improved fit to the data, as compared to the constant dose. Further, the best-fit constant dose in this exercise was 0.35 pg TCDD/kg-day, compared with the current average adult dose of 0.17 pg TCDD/kg-day, as determined by the previous version of this dioxin reassessment (U.S. EPA, 1994b), and with the revised 0.09 pg TCDD/kg-day of this current reassessment. The result provides strong evidence that past doses were, in fact, higher than current doses.

2. The exposure/dose curves in Figure 6-4 suggest that dioxin exposure followed a sharp bell curve, with a precipitous drop to a flat baseline in dose before 1980. This drop is counterintuitive and probably more the result of the simplicity of the pharmacokinetic model than a real-world trend. However, it may be reasonable to treat some generalizations from these calibrated dose curves as reasonable hypotheses. For example, the late 1960s were estimated as years of peak TCDD exposure, an observation that coincides with peaks found in sediment core studies. The estimates derived suggest that TCDD exposures may have been 20 times higher during the 1960s than the 1980s. Over a 10-year peak period in the 1960s and

early 1970s, daily exposures could have been as high as 1.5 to 2.0 pg/kg-day, possibly dropping to as low as 0.10 pg/kg-day and below into the 1980s. Without body burden data, it may be difficult to go much further with the model results.

3. In addition to an exposure dose, the results of this exercise also include temporal body burden levels, as described by body lipid concentrations of TCDD. An example of these results is shown in Figure 6-5. The "specimen year" on the x-axis refers simply to the year in which a cross-section of the population can be examined. For example, in 1986, young individuals have a body burden of about 2 pg/g lipid; whereas older individuals have a body burden exceeding 5 pg/g lipid. The modeled build-up of TCDD in an individual's body can be ascertained by following a curve corresponding to the individual's birth year, shown on the z-axis, and progressing to the left. This figure displays two important trends: (a) body burdens in general tend to be dropping (Schecter, 1991; U.S. EPA, 1991; MAFF, 1995), and (b) body burdens are higher in older individuals than younger individuals (Orban et al., 1994; Andrews et al., 1989; Van der Molen et al., 1996). As seen from specimen years after approximately 1970, body concentrations in individuals of all ages appear to be dropping. At the same time, the cross section of all birth-year populations after about 1970 suggests that concentrations are higher in older individuals. Interestingly, this trend may not have been present in the U.S. population in the mid-1970s and earlier. In the mid-1970s, peak body concentrations appear to have peaked for individuals in their 20s (roughly), with a constant body burden for older individuals. In the 1960s and earlier, differences in body burden do not appear to be a function of age.

4. This model also had a breastmilk feeding component. To search for the best-fit exposure/dose curve, it was arbitrarily assumed that half the population was breast fed and the other half was bottle fed. Therefore, the average concentration for all individuals was calculated as the midpoint of body lipid concentrations modeled with and without breast-feeding. Breast-fed infants were exposed to milk concentrations modeled to occur in 25-year-old females (where breastmilk lipid concentrations were assumed to equal body lipid concentrations). Infants were assumed to be breast fed

for 4 months, and their consumption of breastmilk lipids was 26 g/day. Bottle-fed infants were assumed to be exposed to the general exposure dose, which as can be expected, turned out to be much lower than the breast-feeding dose. Model predictions were compared to a limited subset of the available body burden data, specifically to the under-15 age groups for the two NHATS data sets, where the mean concentrations were 4.2 pg/g lipid (NHATS 1987) and 2.0 pg/g lipid (NHATS 1982). For the under-15 age group in 1982, the predicted mean concentrations in one possible solution were 3.8 pg/g in breast-fed children versus 0.3 pg/g in bottle-fed children; in 1987, the expected means in this age group were 1.8 pg/g for breast-fed and 0.2 pg/g for bottle-fed individuals. Assuming a 50 percent breast-fed rate, predictions yielded averages of 2.0 pg/g and 1.0 pg/g for the 1982 and 1987 NHATS, respectively. Two observations could be made. First, the modeling exercise shows the impact of breast-feeding since modeled predictions of bottle-fed body concentrations were much lower than breast-fed body concentrations (i.e., 0.3 and 0.2 pg/g lipid for bottle fed versus 3.8 and 1.8 pg/g lipid for breast fed). Therefore, if the low body burden found for bottle-fed infants reflects reality, then the NHATS data showing 4.2 and 2.0 pg/mL also show the influence of breast-feeding on the body burden of children. Second, both the model and the data show a drop in the body concentrations of the under-15 age group between 1987 and 1982, suggesting a trend toward declining exposures through the 1980s.

6.8 SUMMARY OF TEMPORAL TRENDS INFORMATION

Some general observations can be made about changes in levels of dioxin-like compounds in the environment over time. These are discussed below and summarized in Table 6-9.

Concentrations of CDD/CDFs and PCBs in the U.S. environment were consistently low prior to the 1930s. Then, concentrations rose steadily until about 1970. At that time, the trend reversed and the concentrations began to decline. That trend has continued to the present. The most compelling supportive evidence of this trend for the CDD/Fs and PCBs comes from dated sediment core studies. Sediment concentrations in these studies are generally assumed to be an indicator of the rate of atmospheric deposition. CDD/F and PCB concentrations in sediments began to increase around the 1930s, and continued to

increase until about 1970. Decreases began in 1970 and have continued to the time of the most recent sediment samples (about 1990). Sediment data from 20 U.S. lakes and rivers from seven separate research efforts consistently support this trend. Additionally, sediment studies in lakes located in several European countries have shown similar trends.

It is reasonable to assume that sediment core trends should be driven by a similar trend in emissions to the environment. The period of increase generally matches the time when a variety of industrial activities began rising and the period of decline appears to correspond with growth in pollution abatement. Many of these abatement efforts should have resulted in decreases in dioxin emissions, i.e. elimination of most open burning, particulate controls on combustors, phase out of leaded gas, and bans on PCBs, 2,4,5-T, hexachlorophene, and restrictions on use of pentachlorophenol. Also, the national source inventory of this assessment documented a significant decline in emissions from the late 1980s to the mid-1990s. Further evidence of a decline in CDD/F levels in recent years is emerging from data, primarily from Europe, showing declines in foods and human tissues.

In addition to the congener specific PCB data discussed earlier, a wealth of data on total PCBs and aroclor mixtures exist which also supports these trends. It is reasonable to assume that the trends for dioxin-like PCBs are similar to those for PCBs as a class because the predominant source of dioxin-like PCBs is the general production of PCBs in aroclor mixtures. PCBs were intentionally manufactured in large quantities from 1929 until production was banned in 1977. U.S. production peaked in 1970, with a volume of 39,000 metric tons. Further support is derived from data showing declining levels of total PCBs in Great Lakes sediments and biota during the 1970s and 1980s. These studies indicate, however, that during the 1990s the decline is slowing and may be leveling off.

Past human exposures to dioxins were most likely higher than current estimates. This is supported by a study which applied a non-steady state pharmacokinetic model to data on background U.S. tissue levels of 2,3,7,8-TCDD from the 1970s and 80s. Various possible intake histories (pg/kg-day over time) were tested to see which best-fit the data. An assumption of a constant dose over time resulted in a poor fit to the data. The “best-fit” (statistically derived) to the data was found when the dose, like the sediment core trends, rose through the 60s into the 70s, and declined to low current levels. Some additional support for this finding comes from a limited study of preserved meat samples from several decades in the twentieth century. One sample, from before 1910, showed

very low concentrations of dioxins and coplanar PCBs. Thirteen other samples, from the 1940s until the early 1980s, consistently showed elevated levels of all dioxin-like compounds as compared to food surveys conducted during the 1990s.

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6-1. Lipid Based Concentrations of CDD/CDFs and PCBs in Samples of Pooled Retail Milk Purchased in the United Kingdom During 1990 and 1995

Location	Concentrations (ppt) TEQ (lipid)			
	CDD/CDFs (1990)[a]	CDD/CDFs (1995)	PCBs (1995)	CDD/CDF/PCBs[b] (1995/1996)
Bristol	2.0	0.92	2.2	3.1
Cambridge	1.4	0.84	1.5	2.3
Carlisle	1.1–1.4	0.67	0.75	1.4
Central London	1.4–3.3	0.99	1.7	2.7
Commuter London[c]	1.7–2.1	0.88	1.7	2.6
Crewe	—	1.4	2.1	3.5
Exeter	1.1–3.3	1.3	1.7	2.9
Northallerton	—	1.1	1.7	2.8
Norwich	—	1.1	2.1	3.2
Nottingham		1.1	2.3	3.5
Slough		0.80	1.6	2.4
Worchester	—	1.0	2.3	3.4

Note: I-TEF_{DF}s were used in calculating I-TEQ_{DF}s; TEF_P-WHO_{94}s were used in calculating TEQ_P-WHO_{94}s.

ppt = parts per trillion

a Some locations were sampled twice in 1990. Samples were also purchased from Beverley, Leeds, and Preston in 1990. Fat contents were not measured for the 1990 samples, and the whole milk concentrations were converted assuming 4 percent fat content, which is typical for UK whole milk.
b The combined concentrations of CDD/CDFs and PCBs were calculated before rounding.
c The individual pints forming this pool were purchased in outer London and the home counties.

Source: MAFF (1997a).

Table 6-2. CDD/F and PCB TEQ Concentrations and Percent Differences from Current TEQ Levels

	Description	I-TEQ_{DF}, pg/g Lipid	TEQ_P-WHO_{94}, pg/g Lipid	Percent of Current I-TEQ_{DF} Levels[a]	Percent of Current TEQ_P-WHO_{94} Levels[a]
1908	Beef ration	0.34 (0.15)	0.07 (0.07)	38 (42)	15 (15)
1945	Beef and pork	0.98 (0.75)	0.36 (0.36)	89 (197)	140 (146)
1957	Dried cream	2.05 (0.81)	3.56 (3.54)	244 (96)	827 (824)
1968	Bacon bar	3.01 (2.94)	1.05 (1.05)	231 (638)	1747 (2620)
1968	Deviled ham	3.73 (3.71)	0.61 (0.61)	287 (805)	1019 (1529)
1971	Beef	1.36 (0.02)	2.48 (1.98)	153 (7)	540 (540)
1971	Bacon wafer	1.75 (1.62)	1.98 (1.98)	135 (352)	3301 (4952)
1977	Raw chicken	1.29 (1.18)	2.72 (2.72)	202 (287)	970 (970)
1977	Cooked chicken	1.33 (1.20)	2.83 (2.83)	209 (292)	1009 (1009)
1979	Pork slices	1.46 (1.20)	0.04 (0.04)	112 (262)	72 (105)
1980	Beef steak	0.94 (0.73)	0.93 (0.93)	106 (207)	203 (203)
1982	Ham slice	1.36 (1.04)	0.07 (0.07)	105 (227)	119 (178)
1983	Beef in bbq	0.50 (0.03)	0.79 (0.79)	56 (8)	171 (171)
1983	Turkey with gravy	0.55 (0.23)	0.32 (0.31)	85 (57)	113 (113)

Note: (results assume ND = ½ LOD; results calculated at ND = 0 shown in parenthesis)

pg/g = picograms per gram

a Current CDD/CDF/PCB levels are the levels observed in national food surveys for beef (Winters et al., 1996a, 1996b), pork (Lorber et al., 1997), poultry (Ferrario et al., 1997), and milk (Lorber et al., 1998).

Source: Winters et al. (1998).

Table 6-3. Estimated Upper Bound Dietary Intakes of CDD/CDFs and PCBs by the Average UK Consumer in 1982 and 1992

Food Group	CDD/CDF Intake (mean) (pg I-TEQ_{DF}/person/day)[a]		PCB Intake (mean) (pg TEQ_P-WHO_{94}/ person/day)[b]	
	1982	1992	1982	1992
Bread	3	4	2	2
Other cereal products	14	17	13	3
Carcass meat	16	4	10	3
Offals (internal organs)	3	1	0.3	0.2
Meat products	15	3	8	3
Poultry	8	2	4	1
Fish	7	3	13	6
Oils and fats	38	6	40	8
Eggs	22	3	5	2
Milk	48	17	28	11
Milk products	66	9	34	7
TOTAL	240	69	156	46

Note: Estimated total dietary intakes were calculated before rounding.

a MAFF (1995).
b Adapted from MAFF (1997b).

Table 6-4. Estimated National Average Concentrations of Dioxins and Furans from the 1982 and 1987 NHATS

Compound	1982[a] (pg/g)	1987[a] (pg/g)	Difference[a] (pg/g)
2,3,7,8-TCDD	5.88 (0.534)	5.38 (0.330)	-0.499 (0.628)
1,2,3,7,8-PeCDD	73.6 (20.0)[b]	10.7 (0.426)	-62.9 (20.0)
HxCDD[c]	122.0 (20.1)[b]	86.8 (3.16)	-34.8 (20.3)
1,2,3,4,6,7,8-HpCDD	142.0 (24.8)	110.0 (3.73)	-31.6 (25.0)
OCDD	768.0 (79.7)	724.0 (28.6)	-43.9 (84.7)
2,3,4,7,8-PeCDF	35.4 (3.57)[b]	9.70 (0.800)	-25.7 (3.66)

a Standard errors of the estimated averages are in parentheses.

b The 1982 estimate is significantly higher than the 1987 estimate at the 0.05 level of significance.

c Analysis results for specific isomers of HxCDD and HxCDF were combined for comparisons.

Source: U.S. EPA (1991).

Table 6-5. Trends in Blood CDD/CDF Levels in a German Population, 1991-1996

Year	1991	1992	1993	1994	1995	1996
Number of samples	95	157	17	74	69	95
Mean age (yrs)	44.7	42.4	40.5	46.5	45.2	37.7
Fat content (mg/g)	5.7	5.7	6.0	6.0	5.7	5.1
Mean total CDD/CDF concentration (pg/g)	718.4	703.2	534.5	376.7	431.6	373.1

Source: Wittsiepe et al. (1998).

Table 6-6. Comparison of Results from the First and Second Round of WHO-Coordinated Human Milk Study

Country	Area	CDDs and CDFs (pg I-TEQ_{DF}/g)				∑ [Marker PCBs] (ng/g)			
		1987/88[a]	n	1992/93	n	1987/88	n	1992/93	n
Austria	Vienna (urban)	17.1	54	10.7	13			381	13
	Tulln (rural)	18.6	51	10.9	21			303	21
Belgium	Brabant Wallou	33.7		20.8	8	558	12	275	8
	Liege	40.2		27.1	20	609	21	306	20
	Brussels	38.8		26.6	6			260	6
Canada	All Provinces 1981			28.6	200			212	200
	All Provinces 1982			14.5	100			112	100
	Maritimes	15.6	19	10.8	20			86	20
	Québec	18.1	34	13.4	20			137	20
	Ontario[b]	17.6	76	18.1	20			128	20
	Prairies	19.4	31	14.6	20			58	20
	British Columbia	23.0	23	15.7	20			70	20
Croatia	Kirk	12.0	14	8.4	10	500[c]	14	218	10
	Zagreb	11.8	41	13.5	13	450[c]	41	219	13
Denmark	Several Regions/Cities	17.8	42	15.2	48	830[c]	10	209	48
Finland	Helsinki	18.0	38	21.5	10	150	38	189	10
	Kuopio	15.5	31	12.0	24	203	31	133	24
Germany	Berlin	32.0	40	16.5	10			375	10
	North Rhine-Westphalia	31.6	79	20.7[e]		762	143		
Hungary	Budapest	9.1	100	8.5	20			61	20
	Scentes	11.3	50	7.8	10			45	10
Netherlands	Rural Area	37.4	13			416	10		
	Urban Area	39.6	13			392	10		
	All Regions	34.2	10	22.4	17	272	96	253	17
Norway[d]	Tromsø (coastal)	18.9	11	10.1	10	562[c]	10	273 (536[c])	10
	Hamar (rural)	15.0	10	9.3	10	507[c]	10	265 (4c83[c])	10
	Skien/Porsgrumm (ind)	19.4	10	12.5	10	533[c]	8	302 (468[c])	10
United Kingdom	Birmingham	37.0		17.9	20			129	20
	Glasgow	29.1		15.2	23			131	23

NOTE: Results are expressed on a fat basis. ∑ (marker PCBs) and I-TEQ_{DF}s are calculated assuming non-detect values are equal to zero.

a Calculated using Nordic TEF-model.
b Ontario-1988 denotes proportional mean of two pooled samples analyzed in the first round.
c Analyzed using packed column technique.
d To compare results between first and second round, samples from 1992/93 have been reanalyzed using (old) packed column technique (Becher and Skåre, personal communication).
e Dioxin levels in human milk samples from North Rhine-Westphalia collected in 1992 as reported by Fürst (1993).

Source: Liem et al. (1996).

Table 6-7. Comparison of CDD/CDF Concentrations in Human Milk from Finland in 1987 and 1992–1994

Selected Congeners	Conc. pg/g fat in 1992-1994		Conc. pg/g fat in 1987	
	Urban Area n = 14	Rural Area n = 28	Urban Area n = 47	Rural Area n = 37
2,3,7,8-TCDF	1.93 ± 0.74***	0.49 ± 0.44	2.98 ± 2.89	6.75 ± 4.29^{xxx}
2,3,7,8-TCDD	2.66 ± 1.46	1.71 ± 0.68	3.37 ± 1.85	2.50 ± 1.25^{x}
2,3,4,7,8-PeCDF	16.3 ± 7.0*	10.4 ± 4.65	20.1 ± 12.5	13.1 ± 5.41
1,2,3,7,8-PeCDD	6.22 ± 2.16*	4.36 ± 1.56	9.78 ± 4.87^{xxx}	7.53 ± 3.23^{xxx}
1,2,3,6,7,8-HxCDD	33.2 ± 8.94	26.9 ± 8.16	48.2 ± 15.8^{xxx}	41.5 ± 15.3^{xxx}
OCDD	230 ± 80.9***	126 ± 55.7	187 ± 83.6	171 ± 71.5^{xxx}
$\sum$CDD/CDF	381 ± 120***	217 ± 76.6	375 ± 132	339 ± 108^{xxx}
I-TEQ_{DF}	19.9 ± 7.42*	13.6 ± 4.57	26.3 ± 11.9	20.1 ± 6.54^{xxx}

Note:

Asterisks indicate a statistically significant difference between urban and rural areas in 1992–1994.
* $p < 0.01$
** $p < 0.005$
*** $p < 0.001$

x indicates a statistically significant difference between 1987 and 1992–1994 results
^{x} $p < 0.01$
^{xx} $p < 0.005$
^{xxx} $p < 0.001$

Source: Kiviranta et al. (1998).

Table 6-8. Mean Human Lipid TCDD Concentrations Reported in Various U.S. Studies

Study (Reference)	Age/Gender Group	Sample Size	Year	TCDD Mean, pg/g	Standard Error of the Mean
Andrews et al. (1989)	18-29, both	14	1986	4.0	0.95
	30-39, both	30	1986	5.9	0.65
	40-49, both	25	1986	5.5	0.71
	50-59, both	22	1986	8.0	0.76
	60-79, both	37	1986	9.5	0.59
Air Force (Michalek et al., 1997)	35-39, male	168	1987	3.8	0.23
	40-44, male	280	1987	4.0	0.18
	45-49, male	165	1987	4.6	0.23
	50-54, male	232	1987	4.7	0.20
	55-59, male	142	1987	4.8	0.25
	60-64, male	33	1987	5.0	0.52
	65-69, male	35	1987	6.2	0.51
NHATS 82 (U.S. EPA, 1991)	0-14, both	178	1982	4.2	0.69
	15-44, both	312	1982	6.9	0.87
	45 + , both	273	1982	5.5	0.84
NHATS 87 (U.S. EPA, 1991)	0-14, both	146	1987	2.0	0.82
	15-44, both	318	1987	4.4	0.52
	45 + , both	401	1987	9.4	0.41
VA/EPA (VA/U.S. EPA,1988)	20-36, male	27	1971–1973	19.8	1.2
	23-39, male	29	1974–1976	17.3	1.2
	26-42, male	57	1977–1979	11.6	1.2
	29-45, male	82	1980–1982	12.6	1.2

Table 6-9. Summary of Findings with Regard to Trends in Dioxin Levels in the Environment and in Humans

Finding	Support	Uncertainty
Concentrations of CDD/CDFs in the environment were consistently low for centuries until the 1930s. Then, concentrations rose steadily until about the 1960s, at which point concentrations began to drop. Evidence suggests that the drop in concentrations is continuing to the present.	***Sediment core studies show a trend of rising concentrations in 1930s and 1940s through the 1960s and 1970s and a subsequent decline to the present.*** - 11 lakes/reservoirs in the U.S.: Cleverly et al., 1996; Versar, 1996 - Lake Huron: Czuczwa et al., 1985a - Green Lake, NY: Smith et al., 1992, 1993 - Hudson River: Smith et al., 1995 - Lakes Superior, Michigan, and Ontario: Pearson et al., 1995 - Straight of Georgia, British Columbia: MacDonald et al., 1992 - A remote arctic lake: Tan et al., 1993; Vartiainen et al., 1995	The assumption of nondegradation of CDD/CDFs in sediment cores.
	Analogous trends in environmental loadings - Rise of the manufacture and use of chlorinated phenolic intermediates and products - Banning of leaded gasoline, certain phenoxy herbicides, PCBs - Reductions in pulp and paper mill discharges - EPA National Source Inventory showing 60% reduction in CDD/CDF TEQ emissions between 1987 and 1995 (see Volume II)	Indirect measure of environmental levels.
	Limited trend for other environmental concentrations - Rises and declines in historical food products in the U.S.: Winters et al., 1998 - Rises and declines in herbage, soil, and air measured in archived samples in UK: Kjeller et al., 1991, 1996; Harner et al., 1995 - Reductions in the past two decades in herring gull eggs in the Great Lakes and the Gulf of St. Lawrence River (Hebert et al., 1994); pike in Sweden (DeWit et al., 1994); pike in Finland (Korhonen et al., 1995); air in Germany (Hiester et al., 1995); German dairy products and human milk between 1990 and 1994 (Fürst and Wilmers, 1995; 1997)	Very few archived environmental measurements to ascertain trends beyond the past decade or so. More recent data showing a decline in trends is limited.
	Suggestive evidence of declines in human body burdens in recent decade - National Human Adipose Tissue Survey: U.S. EPA, 1991 - Ministry of Agriculture, Fisheries, and Food's calculations of declines in dose based on market basket surveys showing reductions in levels in combination with reductions in consumption of key food items: MAFF (1995)	Long half-lives in humans impede responses in body burdens to changes in environmental levels.
Environmental levels of coplanar PCBs began increasing in the 1920s, peaking in the 1970s, and decreasing after that	***PCB production data*** - Rise in the manufacture from the latter 1920s to the early 1970s; complete ban in 1977	Nearly all trends data are specific to PCBs or Aroclors, and not to coplanar PCBs.

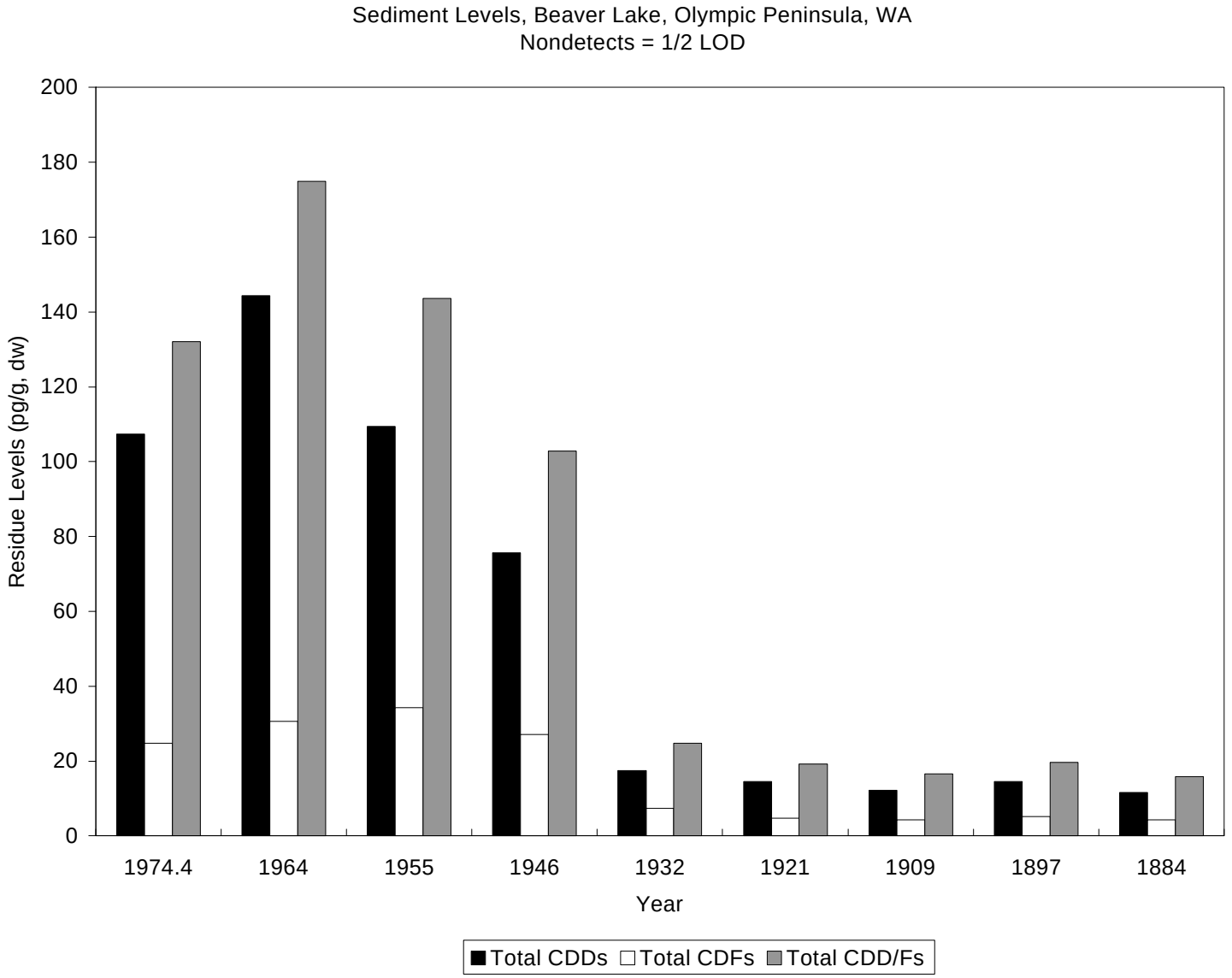

Figure 6-1. CDD/CDF Levels in Sediment, Beaver Lake, Washington

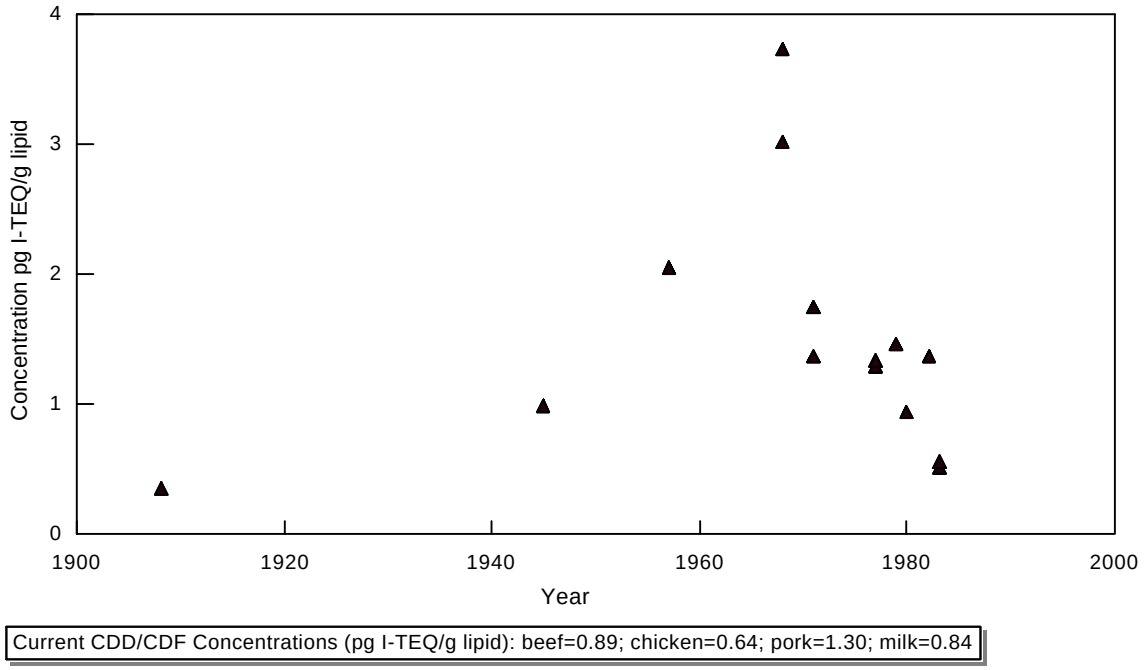

Figure 6-2. $I\text{-}TEQ_{DF}$ Concentrations of Historical Food Samples from the U.S. (results calculated at ND = ½ LOD)

Source: Adapted from Winters et al. (1998).

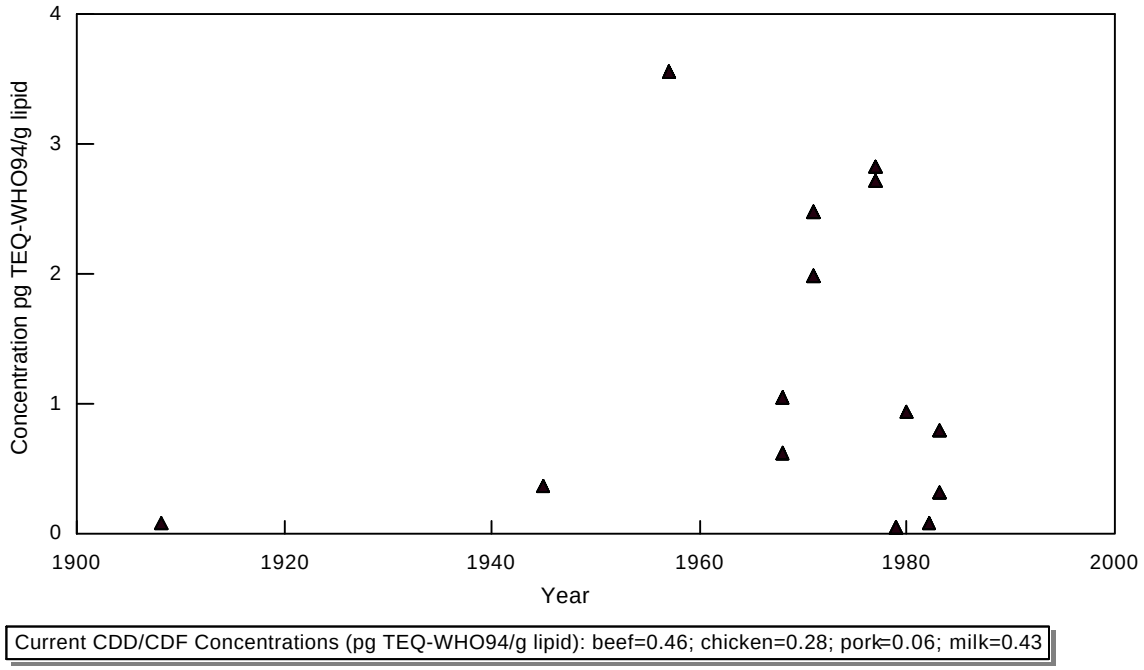

Figure 6-3. $TEQ_P\text{-}WHO_{94}$ Concentrations of Historical Food Samples from the U.S. (results calculated at ND = ½ LOD)

Source: Adapted from Winters et al. (1998).

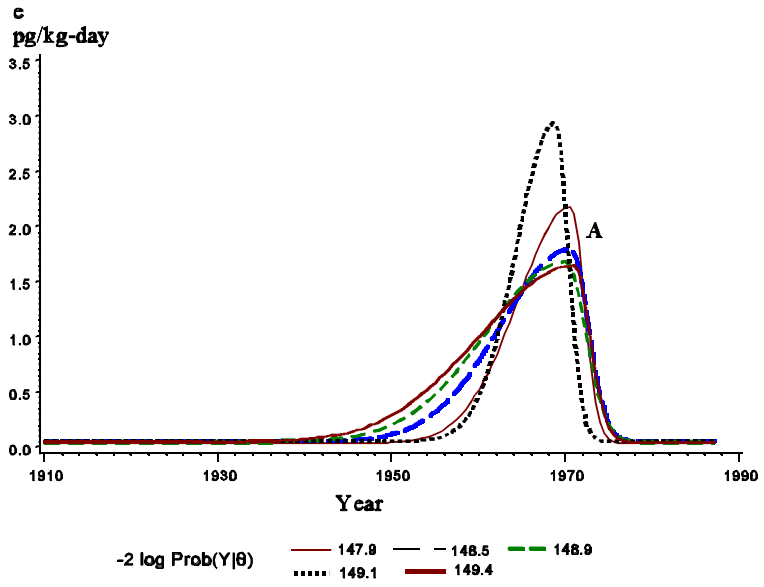

Figure 6-4. Examples of Temporal Exposure Curves for 2,3,7,8-TCDD, e(t) in Units of pg/kg-day.

Source: Pinsky and Lorber (1998).

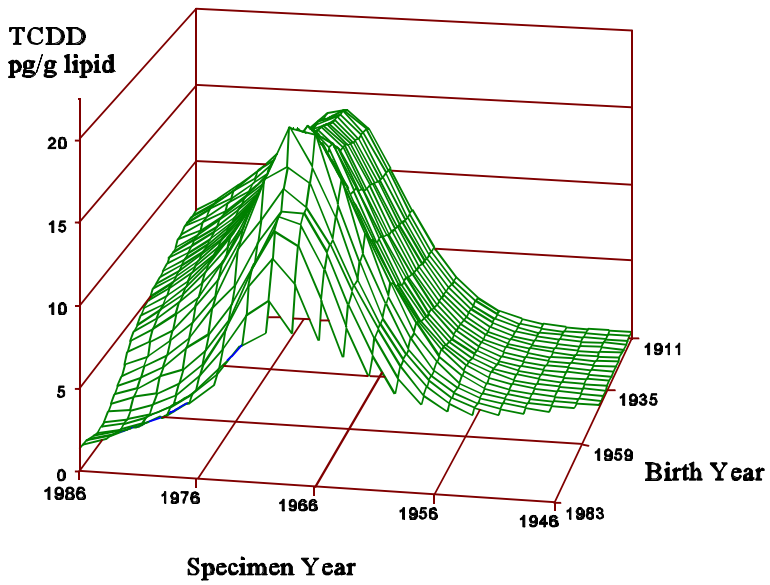

Figure 6-5. Predicted Mean TCDD Lipid Concentrations (pg/g) in Males by Birth Year and Specimen Year Derived Using e(t) Curve Labeled A in Figure 6-4

Source: Pinsky and Lorber (1998).